ABSTRACTS OF CARDIOLOGY

Edited by Dr. J. L. Lovibond in collaboration with Abstracts of World Medicine

Peripheral Aneurysm and Arteriovenous Fistula. S. M. COHEN. *Ann. Roy. Coll. Surg. Engl.*, **11**, 1–30, July, 1952.

His experience in a centre for vascular diseases during the last war is the basis of this article, in which the author suggests an explanation of behaviour of the circulation in aneurysm and arterio-venous fistula. Pulsatile stretching is the stimulus for the production of elastic tissue; the thickening of a vein grafted into an artery, or of one taking part in an arterio-venous fistula are examples of this process. Contrariwise, the thinned, dilated afferent arterial trunk to such a fistula undergoes these alterations because of the loss of pulse pressure resulting from the fistula. The number and tortuosity of the collateral channels that develop after occlusion of a main artery depend upon the peripheral resistance in the distal segment. If this is healthy, resistance is low and the by-passing vessels are short and direct; they share with the normal artery, which they bridge, a pulsatile content. If pulsation be restored to the dilated artery leading to a fistula by ligation, shortening and reduction in diameter follow. Post-stenotic aneurysms are due to loss of the normal impulse beyond the obstruction.

C. G. Rob

Anæsthesia and the Cardiac Patient. G. W. HAYWARD. *Anæsthesia*, **7**, 67–71, April, 1952.

The author assesses the operative risks of major surgery in patients with heart disease as being only a little higher than in those without heart disease provided full advantage is taken of up-to-date surgical and anæsthetic techniques. He regards two factors as of especial importance—anoxia and hypotension. He considers that sudden death is most likely to occur in patients with coronary disease, aortic stenosis, syphilitic aortitis, heart-block, or a large patent ductus arteriosus; "death is usually the result of ventricular fibrillation in these patients." Preoperative treatment with procaine amide hydrochloride (pronestyl) is advised in a dosage of 500 to 1000 mg. orally; if cardiac irregularity occurs during operation the drug may be given intravenously at a rate not exceeding 100 mg. per minute up to a total dose of 1000 mg.

It is pointed out that controlled hypotension is regarded by cardiologists as particularly dangerous in elderly arteriosclerotic patients or in those with coronary disease, but that too little is known about the effects of this technique for a balanced opinion to be given. It is stated that whereas hypotension can be avoided in those with a history of angina pectoris or other suggestive signs of coronary impairment, it is not uncommon for coronary insufficiency to be silent until myocardial ischæmia occurs during operation, and there is no reliable way of detecting it pre-operatively. In view of such unpredictable risks it would therefore seem wise at present to restrict the use of controlled hypotension to operations where success depends on a bloodless operative field, thus justifying acceptance of the increased risks involved.

It is recommended that in congestive heart failure operation be postponed until the heart rate has been stabilized, and pulmonary congestion should be counteracted by careful control over the fluid balance and the use of mercurial diuretics. As the author says, "one method of treating or preventing attacks of acute pulmonary œdema in man is the administration of a rapidly acting barbiturate."

Cardiac arrhythmias are not considered to call for specific measures beyond normal care; and it is suggested that angiocardiography is best performed under basal narcosis in adults, but under general anæsthesia in children.

[The author is to be congratulated on a very concise review of the subject which is a valuable and practical contribution.] *Michael Kerr*

Prognosis of Angina Pectoris. Observations in 6882 Cases. W. J. BLOCK, E. L. CRUMPACKER, T. J. DRY, and R. P. GAGE. *J. Amer. med. Ass.*, **150**, 259–264, Sept. 27, 1952.

The case histories of 6882 patients suffering from angina pectoris associated with coronary sclerosis who were examined at the Mayo Clinic over a period of 18 years have been analysed. The minimum follow-up period was 5 years and the maximum 23 years.

The average age at diagnosis in the Clinic was 58·8 years, at which time the average duration of the angina was 2·5 years. Mortality was greatest in the first year (about 15%) and was about 9% annually thereafter. The 5-year survival rate for the entire series was 58·4%, compared with 86·9% for the normal population. The 10-year survival rate was 37·1%, compared with the normal rate of 70·4%. Patients in the younger age groups showed higher survival rates at the 5- and 10-year periods than the older patients, but in each group the rate bore about the same relation to the normal. The prognosis was better in women, with a 10-year survival rate of 49%, than in men.

The presence of cardiac enlargement, hypertension, myocardial infarction, and congestive failure influenced the prognosis unfavourably. Electrocardiographic findings were of definite prognostic value. Thus the 10-year survival rate for patients with normal tracings when first seen was 50%, for those with Q- or T-wave changes in

the electrocardiogram 24%, for those with evidence of left bundle-branch block 14%, and for those with auricular fibrillation 13·5%. The 10-year survival rate for patients in whom angina was associated with obesity or gall-bladder disease was 44%, with thyroid disease 37%, with diabetes 21%, and with carcinoma 17%. The reason for the more favourable outlook in the first group is not clear.

T. Semple

An Evaluation of Anticoagulant Therapy in the Treatment of Acute Myocardial Infarction. H. I. RUSSEK and B. L. ZOHMAN. *Amer. Heart J.*, **43**, 871–880, June, 1952.

At the State University of New York College of Medicine 1047 patients suffering from myocardial infarct were examined and classified as " good " or " bad " risks on the day of admission to hospital; in 60% of cases admission took place within 24 hours of the attack. All those revealing a previous myocardial infarct, intractable pain, persistent or severe shock, enlarged heart, congestive failure, gallop rhythm, fibrillation, flutter, tachycardia or block, varices, and diabetic acidosis were classified as bad risks. Conservative treatment without the use of anticoagulants was instituted. The crude mortality rate was 33%, 3% in the former group and 60% in the latter. Similarly the incidence of thrombo-embolic complications in the survivors was 0·8% in the " good " group and 10% in the " bad " group. It is concluded that the use of anticoagulants is not justified in " good risk " cases of infarct, especially as most of the cases that died in this group could not have been affected by such treatment, as death was not primarily due to thrombosis.

No correlation was found between age and prognosis; the division into two groups as a result of clinical findings was all-important in prognosis. It follows, therefore, that the decision as to whether or not anti-coagulants are used depends on the findings in the individual case, only the " bad risks " being given this treatment. The replies to a questionary issued to American consultant physicians indicated that about one-half of them use anticoagulants routinely, partly as a result of pressure from patients or their relatives, and from practitioners. Not unexpectedly, some 45% of the consultants had encountered serious hæmorrhagic complications. The routine use of anticoagulant therapy is deprecated by the authors.

James D. P. Graham

Factors Influencing Rupture of the Heart After Myocardial Infarction. R. W. OBLATH, D. C. LEVINSON, and G. C. GRIFFITH. *J. Amer. med. Ass.*, **149**, 1276–1281, Aug. 2, 1952.

The authors review the records of 80 cases of perforation of the heart following myocardial infarction which have occurred at the Los Angeles County Hospital in the last 10 years. From this study he following points emerge: the mean age incidence was about 70 years, none of the patients being under 50; slightly more than half the patients were women; cardiac rupture occurred in 7% of all hospital cases of myocardial infarctions.

The complication seemed to be unrelated to the interval elapsing before the institution of treatment, for four-fifths of the cases were admitted within 72 hours of onset. There was no evidence that ordinary activity predisposed to rupture. In about half the cases rupture occurred within 72 hours and in 14 cases in less than 24 hours; in 6 cases intervals of from 19 to 30 days were noted. A QS pattern in the electrocardiogram was often seen and in the opinion of the authors would have been found in all cases had sufficient leads been taken. Persistent hypertension after infarction was the rule. No evidence was found to incriminate digitalis or other drugs.

At necropsy the striking feature was the close relationship of the site of perforation to the interventricular septum, being in, parallel to, or adjacent to that structure in 67% of cases. The great majority of perforations were dissections by devious routes from the endocardium to the pericardium; direct rupture was rare.

H. David Friedberg

The Routine Use of Quinidine in Acute Myocardial Infarction. F. B. CUTTS and B. RAPOPORT. *New. Engl. J. Med.*, **247**, 81–83, July 17, 1952.

The authors studied the effect of quinidine on the incidence of arrhythmia and of sudden, unexpected death in cases of myocardial infarction treated with dicoumarol. The patients, in all of whom there was clear electrocardiographic evidence of recent infarction, were divided into 3 groups. In the first group 66 patients received 0·2 g. of quinidine orally 4 times a day; in the second group 20 patients received 0·4 g. every 8 hours; in the third group 25 patients received 0·6 g. every 8 hours. Controls numbered 55 in the first group, 20 in the second, and 25 in the third.

The incidence of extrasystoles was the same in all groups. Auricular fibrillation and flutter occurred in some cases in each group; the beneficial effect of quinidine in preventing these irregularities was difficult to prove and, at best, was only moderate. There was no evidence that quinidine either prevented or promoted auriculoventricular block. No significant difference in mortality between the groups was noted. The incidence of sudden death (excluding death from myocardial rupture) was least in the group receiving 0·2-g. doses, and it is suggested that the heavier dosage may have been responsible for some of the deaths. It is concluded that there is no indication for the routine use of quinidine in acute myocardial infarction. *R. S. Stevens*

The Reversibility of Malignant Hypertension. G. W. PICKERING, A. DICKSON WRIGHT, and R. H. HEPTINSTALL. *Lancet*, **2**, 952–956, Nov. 15, 1952.

Essential hypertension behaves in one of two ways: in the majority of patients it pursues a relatively benign course over many years, to terminate in heart failure, apoplexy, or intercurrent disease; in a few patients, usually younger, it follows a rapidly fatal course, with albuminuric retinitis and progressive renal failure terminating in uræmia. It has been suggested that this latter malignant type is an expression of the severity of the hypertensive process and occurs if the arterial pressure

rises and is maintained above a certain critical level so that arteriolar necroses occur; and further, that if this sustained pressure can be reduced sufficiently and for long enough the malignant form of the disease should revert to the benign form.

The present authors describe the clinical findings in 3 cases of malignant hypertension, with arteriolar necroses in the kidney and adrenal glands, and albuminuric retinitis. Arterial pressure was reduced by nephrectomy in one patient, by subtotal adrenalectomy in the second, and by sympathectomy and subtotal adrenalectomy in the third. As a result the 3 patients remain alive and well 5 years after operation, with less severe hypertension normal blood urea level, and normal urea concentration.

It is concluded that malignant hypertension is reversible, and that the benign and malignant forms are the simple consequences of the severity of the hypertension.

James W Brown

Prolongation of Action of Hypotensive Drugs. Use of Polyvinylpyrrolidone and Dextrans to Lengthen Effects of Hexamethonium and Hexamethylene Bisethyldimethylammonium. F. H. SMIRK. *Lancet*, **2**, 695–699, Oct. 11, 1952.

By incorporating either poly-vinyl-pyrrolidone or dextran in the solution for subcutaneous injection of methonium drugs absorption of the latter is delayed and their hypotensive action is prolonged. The duration of their action can be further prolonged, so that they become significantly effective for a full 5 to 8 hours, by the addition of a local vasoconstrictor such as ephedrine hydrochloride. It is usually necessary to increase the dose of the methonium compound by at least 30% when changing from a simple aqueous solution to the slowly-absorbed preparation. Oral administration of hexamethonium bromide is useful when the blood pressure begins to rise again after injection of the slow-absorption preparation, steady control being possible by this method equivalent to three subcutaneous injections of the simple aqueous solution. *J. L. Lovibond*

High Blood Pressure in the Elderly. H. DROLLER, J. PEMBERTON, C. ROSEMAN, and J. L. A. GROUT. *Brit. med. J.*, **2**, 968–970, Nov. 1, 1952.

From a random sample, taken from the Sheffield food office register, of people of pensionable age, a group of 476, who were living at home, either alone or alone with spouse, were medically examined, and the incidence among them of arterial hypertension, with reference to its association with other signs and symptoms, was analysed. It was found that 57% of the men and 77% of the women had a systolic pressure of 160 mm. Hg or more, and 28% and 48% respectively had a diastolic pressure of 100 mm. Hg or more. No case of malignant hypertension was found, all the cases of hypertension being regarded as benign. The difference in blood pressure between certain age groups was not significant.

The presence of vertigo or of tinnitus could not be related to the blood pressure level, nor could the presence of angina of effort be related to the degree of hypertension. The incidence of arteriosclerosis increased significantly with age, but the degree of arteriosclerosis was not related to the degree of hypertension. Arteriosclerosis was, in fact, commoner in men than women, while hypertension was commoner in women. There was no significant association between the blood pressure level and the size of the heart, estimated from a chest radiograph. *J. C. Brocklehurst*

Commissurotomy for Mitral Stenosis (Results and Indications for Operation). P. SOULIÉ, J. DI MATTÉO, R. TRICOT, and L. MOREAU. *Bull. Soc. méd. Hôp. Paris*, **68**, 871–894, 1952.

Since September, 1950, the authors have performed mitral valvotomy in 52 out of 55 cases operated upon; there were 5 postoperative deaths, 3 of which occurred in long-standing advanced cases. Of the 52 patients, 35 have been followed up for 3 months or more, and of these about three-quarters were greatly improved by the operation, while in one-quarter there was little or no improvement.

The selection of patients and their investigations is described in detail. The authors affirm that the ideal indications for the operation are: an uncomplicated mitral stenosis occurring in a patient between 25 and 35 years of age, with marked functional difficulties (such as recurrent pulmonary œdema, dyspnœa on effort, and hæmoptysis), no notable enlargement of the heart volume, electrocardiographic signs of overloading of the right ventricle, increased pressure in the pulmonary circulation, and a diminution of the mitral surface to less than 1·5 sq. cm. The results of cardiac catheterization are of particular interest in those cases in which there is an unusually high gradient between pulmonary artery pressure and capillary pressure. This is apparently due to a raised pulmonary peripheral resistance other than the passive hypertension caused by the mitral stenosis. In keeping with these findings pulmonary atheromatous changes were found at necropsy in 3 of their cases. *S. F. Stephenson*

The Diagnosis of Mitral Incompetence accompanying Mitral Stenosis. Review of Eleven Cases Treated Surgically. A. LOGAN and R. TURNER. *Lancet*, **2**, 593–598, Sept. 27, 1952.

The authors, working in Edinburgh, have studied 60 patients who have undergone a thoracotomy for mitral disease. They consider that the classical signs of mitral incompetence (apical systolic murmur; enlargement, often with systolic expansion, of the left atrium; and clinical, radiological, or electrocardiographic evidence of left ventricular hypertrophy) are not present in all cases. Even in the presence of a murmur of mitral stenosis, predominant incompetence should be suspected if the first heart sound at the apex is absent or faint and if the opening snap of the mitral valve is not heard. The left atrium is usually considerably enlarged if there is any significant incompetence. Systolic expansion of the left atrium is a reliable sign only if it is gross. " Systolic expansion " has been observed preoperatively in patients in whom no regurgitation has been felt at operation.

In these cases it is probable that the appearance of systolic expansion has been due to backward displacement of the whole atrium. The detection of hypertrophy or dilatation of the left ventricle is important in a patient with mitral stenosis, since it signifies some complicating lesion—usually systemic hypertension, aortic valvular disease, or mitral incompetence. If, as is usual, hypertension and aortic valvular disease can be excluded, even slight ventricular enlargement indicates mitral incompetence.

A series of 11 patients suffering from mitral incompetence have been treated surgically by placing a pedicled sheet of pericardium across the ventricular surface of the mitral valve. In 4 cases there was also a stenosis, requiring a preliminary valvulotomy. The breadth of the regurgitant stream felt on the intracardiac finger was generally more significant than its force. A 7-in. (17·5 cm.) silver probe was passed eye first through the ventricle from front to back, and then threaded with a sheet of pericardium pedicled on the diaphragm opposite the auriculo-ventricular ring. The probe was withdrawn, followed by the graft, which was then sutured to the ventricular wall. The position and tension of the graft were ascertained by palpation from within the atrium through the mitral valve. The point of entry of the probe was in the angle between the left coronary artery and its interventricular branch, 2 cm. from each vessel. The point of exit was similarly related to the left coronary artery and the interventricular branch of the right coronary artery.

Although 10 patients have been improved (3 to 10 months after operation), no final assessment of the value of the operation can yet be made. There have been no deaths. *R. L. Hurt*

The Left Auricular Pressure Pulse in Normals and in Mitral Valve Disease. A. WYNN, M. B. MATTHEWS, I. K. R. MCMILLAN, and R. DALEY. *Lancet*, **2**, 216–219, Aug. 2, 1952.

At St. Thomas's Hospital, London, the pressure in the left auricle was recorded during thoracotomy in 14 patients with normal rhythm, in 19 patients with mitral stenosis and normal rhythm, and in 18 patients with mitral stenosis and auricular fibrillation. In some patients the left auricular pulse was also recorded after valvotomy. In patients with normal hearts the first event is a wave due to auricular systole varying from 2 to 8 mm. Hg in amplitude; this is followed rapidly by a further rise in pressure beginning at the end of the QRS complex, but this deflection may be slight or absent. There is then a marked fall in auricular pressure followed by a rise reaching its peak (1 to 9 mm. Hg) 0·04 to 0·12 second after the end of the T wave. Then follows a sharp fall in pressure at the opening of the mitral valve, which is followed by a slight rise before the next auricular systole.

In a few patients tracings were obtained from the right auricle and these were similar in form to those from the left. In 5 patients with mitral stenosis significant mitral incompetence was found at operation. There was no striking difference in tracings of the left auricular pressure in these patients from those from patients with no mitral incompetence. But in one patient with preoperative mitral incompetence the pressure in late systole rose to 25 mm. Hg. It is concluded that mitral regurgitation in the presence of mitral stenosis does not produce a qualitative change in the form of the left auricular pressure tracing. *C. Bruce Perry*

The Role of the Bronchial Veins in Mitral Stenosis. J. C. GILROY, P. MARCHAND, and V. H. WILSON. *Lancet*, **2**, 957–959, Nov. 15, 1952.

Two distinct systems of bronchial veins may be distinguished: (1) the true bronchial veins, which drain the intrapulmonary bronchi and the interstitial framework of the lung; and (2) the pleurohilar veins, which drain the hilar structures and the subpleural planes of the lung. The true bronchial veins drain into the pulmonary veins, whereas the pleurohilar veins empty into systemic veins, such as the azygos or intercostal veins. The pleurohilar veins communicate freely with the pulmonary veins at the hilum and in the subpleural plane.

Necropsy in 6 cases of mitral stenosis revealed congestion and dilatation of the pleurohilar veins, which were shown to communicate freely with the pulmonary veins and drained directly into the azygos and hemiazygos veins. These findings conform with the observation of grossly congested hilar veins at operation. Dilitation of the pleurohilar veins suggests a mechanism whereby the rise in pressure in the pulmonary veins may be modified and considerable amounts of blood take part in a circus movement through them from the right ventricle to the right auricle without traversing the left side of the heart. It is suggested that the "stiff lung" of mitral stenosis is explained by a rise in bronchial venous pressure, which may ultimately cause a transudation of œdema fluid into the supporting interstitial framework of the lung, thus causing rigidity without any intra-alveolar pulmonary œdema.

James W. Brown

The Relapsing Form of Acute Benign Pericarditis of Unknown Ætiology. B. COBLENTZ, J. L. FUNCK-BRENTANO, and J. LENÈGRE. *Sem. Hôp. Paris*, **28**, 2923–2931, Oct. 2, 1952.

The authors describe 3 cases of pericarditis in which the disease lasted about 6 weeks but relapsed from 3 to 5 times over the course of the next 6 months. No definite ætiology could be determined in these patients, whose ages ranged from 33 to 46 years. The literature of acute benign pericarditis is reviewed and from this it is noted that relapses occurred in 15% of the cases.

G. S. Crockett

Epidemic Non-specific Pericarditis. S. AAGAARD and S. E. JENSEN. *Nord. Med.*, **48**, 1409–1411, Oct. 10, 1952.

From the Central Hospital, Randers, Denmark, 7 cases of acute non-specific pericarditis are described. The age of the patients ranged from 11 to 36 years. The febrile period lasted for 3 to 21 days, and pericardial friction

was observed for periods up to 17 days. There were no signs of cardiac inefficiency, but a shock-like condition with tachycardia and hypotension was seen in some patients. Rheumatic fever was excluded in all cases. The patients were treated with ACTH, 40 mg. daily for 10 days; some subjective improvement was noted. Five of the cases occurred during an epidemic of Bornholm disease, and it is suggested that the two conditions may be associated. *D. J. Bauer*

Lesions in Auricular Appendages Removed at Operations for Mitral Stenosis of Presumed Rheumatic Origin. D. C. SABISTON and R. H. FOLLIS. *Bull. Johns Hopk. Hosp.*, **91**, 178–182, Sept., 1952.

The authors' description of the histological changes in the left auricular appendage removed at operation for mitral commissurotomy is based on 43 specimens removed by Blalock from patients with " pure " mitral stenosis, the ages of the patients ranging from 21 to 48 years. These histological changes are classified and illustrated. Rheumatic lesions are defined as Aschoff bodies or cellular accumulations leading thereto. The frequency and extent of the rheumatic lesions were not related to a history of rheumatic fever, or to the presence of fibrillation, or to age. There was some correlation with sedimentation rate, since all those with a raised sedimentation rate had rheumatic lesions.

E. G. L. Bywaters

Significance of Pulmonary Hypertension in Constrictive Pericarditis. A Pre- and Post-operative Study. J. G. SCANNELL, G. S. MYERS, and A. L. FRIEDLICH. *Surgery*, **32**, 184–189, Aug., 1952.

Cardiac catheterization was carried out before and after operation in 5 cases in which pulmonary hypertension was a constant feature of constrictive pericarditis. The authors found that there was no obstruction to the entrance of blood into the right auricle, but there was absence of filling of the ventricle during diastole. They noted a persistent rise in the pulmonary artery pressure and considered that the right and left sides of the heart were equally affected by the disease process. After operation the pulmonary arterial pressure remained significantly raised in 3 of the 5 patients, and in one it actually rose, although in all the pressure in the right side of the heart fell.

The importance of the release of both the left and the right ventricles at operation is emphasized.

J. R. Belcher

Surgical Closure of Defects of the Interauricular Septum by the Use of an Atrial Well. R. F. GROSS, A. A. POMERANZ, E. WATKINS, and E. I. GOLDSMITH. *New Engl. J. Med.*, **247**, 455–460, Sept. 25, 1952.

The authors have investigated the problem of the treatment of intra-atrial septal defects. They discuss the various methods by which attempts have been made to treat this lesion, and find them all open to criticism.

By means of extensive experiments on dogs they have developed a technique that employs an " atrial well," that is, a conical rubber funnel which is sewn to the atrial wall, and into which blood is allowed to flow from the open heart chamber until it reaches the level of mean intra-atrial pressure. Through the orifice at the lower end it has been possible to undertake deliberate intracardiac manœuvres without thrombosis in the well or interference with the circulation. Various methods for occluding the defect were tried, and are described. The authors have decided that where an orifice is 1 cm. or less in diameter it can safely be sutured, but where it is larger than this a plastic sheet should be sutured into place.

Using this technique the authors have now operated on 6 patients with intra-atrial septal defects. For use in human subjects they used a " well " 15 cm. in length with an upper orifice diameter of 13 cm., and a lower orifice diameter of 4 cm., capable of being expanded to 6 cm. by a special self-retaining retractor. Details of the cardiac lesion in the 6 cases are given. The first 4 patients died, but the last 2 survived and were very much improved, and it is felt that the operation may, in future, carry a low mortality. *J. R. Belcher*

Pericardial Cysts. W. M. LOEHR. *Amer. J. Roentgenol*, **68**, 584–609, Oct., 1952.

In this long article the author correlates previous reports on pericardial cysts, presents a classification, discusses the radiological aspects, and reports on 6 personal cases. The pathology of the various types is also described.

In discussing radiological diagnosis the author lays stress on fluoroscopic examination and spot films, and particular attention should be paid to alteration in size of the opacity with change in posture. The differential diagnosis is discussed; although certain features are helpful, diagnosis is still rather uncertain, and the decision as to whether operation is justified may be a difficult one. *Sidney J. Hinds*

Influence of Penta-erythritol Tetranitrate (Peritrate) on Acute and Chronic Coronary Insufficiency. T. WINSOR and P. HUMPHREYS. *Angiology*, **3**, 1–15, Feb., 1952.

Penta-erythritol tetranitrate, a white crystalline drug, was administered orally in doses of 1 to 6 10-mg. tablets daily to 125 patients with angina pectoris and an equal number with other forms of chest pain.

The patients recorded the number of attacks daily for 3 months while taking the tablets and for 3 months without medication, 80% of those with angina showing a satisfactory reduction in the number of attacks, whereas only 4% of the control group were improved. In 30 patients the effect of peritrate was compared with those of aminophylline, khellin, and theobromine calcium salicylate. The frequency of attacks during treatment with these drugs was about the same as when a placebo was given, but was reduced to one-quarter with peritrate. Exercise tolerance in angina pectoris was improved by 50% by peritrate, the effect beginning 1½ hours after administration and continuing for about 5 hours.

C. W. C. Bain